WE CLAIM:

1. A method of modulating inflammation within an immune privileged site in an animal by introducing an effective amount of a Fas ligand fragment comprising the extracellular domain of a full length Fas ligand, or a derivative thereof, behind the blood-tissue barrier of the immune privileged site, wherein said Fas ligand fragment, or derivative thereof, has the ability to induce apoptosis in Fas expressing cells.

2. The method according to claim 1, wherein said immune privileged site is selected from the group comprising: the central nervous system (CNS); eye; placenta; testis; and ovaries.

3. The method according to claim 1, wherein said effective amount of the Fas ligand fragment, or derivative thereof, is administered to said animal by a method selected from the group comprising: intrathecal administration; intraventricular administration; and intracisternal administration.

4. The method according to claim 1, wherein said Fas ligand fragment is a recombinant polypeptide.

5. The method according to claim 1, wherein said Fas ligand fragment comprises at least amino acids 103-281 of a human full length Fas ligand.

6. The method according to claim 2, wherein said immune privileged site is the CNS.

7. The method according to claim 6, wherein said inflammation is associated with an autoimmune disorder.

8. The method according to claim 7, wherein said autoimmune disorder is multiple sclerosis.

9. The method according to claim 7, wherein said autoimmune disorder is experimental allergic encephalomyelitis (EAE).

10. The method according to claim 6, wherein said inflammation is associated with a disorder selected from the group comprising: optic neuritis; Devic's disease; encephalitis; myelitis; encephalomyelitis; acute disseminated encephalomyelitis; acute necrotizing hemorrhagic leukoencephalomyelitis; acute transverse myelitis; limbic encephalitis; post-polio syndrome; subacute sclerosing panencephalitis; Guillian-Barre syndrome; acute, subacute, and chronic neuropathy, in which there is radiculitis within the spinal canal; aseptic meningitis; chronic and recurrent meningitis; stroke; CNS trauma; CNS compression; infection; psychiatric diseases; inflammation or rejection after CNS transplantation; neurodenerative diseases; Alzheimer's disease; Parkinson's disease; Huntington's disease; amyotrophic lateral sclerosis; HIV-related encephalopathy; and "stiff-man" syndrome.

11. The method according to claim 2, wherein said immune privileged site is the eye.

12. The method according to claim 11, wherein said inflammation is associated with a disorder selected from the group comprising: uveitis; conjunctivitis; chorioretinitis; uveoretinitis; optic neuritis; intraocular inflammation, such as retinitis and cystoid macular edema; sympathetic ophthalmia; scleritis; retinitis pigmentosa; inflammatory components of degenerative fondus disease; inflammatory components of ocular trauma; ocular inflammation caused by infection; proliferative vitreoretinopathies; acute ischemic optic neuropathy; excessive scarring, for example, following glaucoma filtration operation; and inflammation reaction against ocular implants.

13. The method according to claim 2, wherein said immune privileged site is the testis.

14. The method according to claim 13, wherein said inflammation is associated with a disorder selected from the group comprising: orchitis; epididimo-orchitis; infertility; and orchidal trauma.

15. The method according to claim 1, wherein said animal is a mammal.

16. The method according to claim 15, wherein said mammal is a human.

17. A method of creating an immune privileged site in a tissue of an animal comprising administering an effective amount of Fas ligand fragment comprising the extracellular domain of a full length Fas ligand, or a derivative thereof, behind the blood-tissue barrier of the tissue, wherein said Fas ligand fragment, or derivative thereref, has the ability to induce apoptosis in Fas expressing cells.

18. The method according to claim 17, wherein said animal is a mammal.

19. The method according to claim 18, wherein said mammal is a human.

20. A method of modulating inflammation in an immune privileged site in an animal through the *in vivo* induction of apoptosis in Fas expressing cells, comprising introducing an effective amount of a Fas ligand fragment comprising the extracellular domain of a full length Fas ligand, or a derivative thereof, behind the blood-tissue barrier of the immune privileged site.

21. The method according to claim 20, wherein said animal is a mammal.

22. The method according to claim 21, wherein said mammal is a human.

23. A method of modulating inflammation within an immune privileged site in an animal by introducing an effective amount of a nucleic acid expressing a Fas ligand fragment comprising the extracellular domain of a full length Fas ligand, behind the blood-tissue barrier of the immune privileged site, wherein said Fas ligand fragment, or derivative thereof, has the ability to induce apoptosis in Fas expressing cells.

24. The method according to claim 23, wherein said immune privileged site is selected from the group comprising: the central nervous system (CNS); eye; placenta; testis; and ovaries.

25. The method according to claim 23, wherein said effective amount of the Fas ligand fragment, or derivative thereof, is administered to said animal by a method selected from the group comprising: intrathecal administration; intraventricular administration; and intracisternal administration.

26. The method according to claim 23, wherein said Fas ligand fragment is a recombinant polypeptide.

27. The method according to claim 23, wherein said Fas ligand fragment comprises at least amino acids 103-281 of a human full length Fas ligand.

28. The method according to claim 24, wherein said immune privileged site is the CNS.

29. The method according to claim 28, wherein said inflammation is associated with an autoimmune disorder.

30 The method according to claim 29, wherein said autoimmune disorder is multiple sclerosis.

31. The method according to claim 29, wherein said autoimmune disorder is experimental allergic encephalomyelitis (EAE).

32. The method according to claim 28, wherein said inflammation is associated with a disorder selected from the group comprising: optic neuritis; Devic's disease; encephalitis; myelitis; encephalomyelitis; acute disseminated encephalomyelitis; acute necrotizing hemorrhagic leukoencephalomyelitis; acute transverse myelitis; limbic encephalitis; post-polio syndrome; subacute sclerosing panencephalitis; Guillian-Barre syndrome; acute, subacute, and chronic neuropathy, in which there is radiculitis within the spinal canal; aseptic meningitis; chronic and recurrent meningitis; stroke; CNS trauma; CNS compression; infection; psychiatric diseases; inflammation or rejection after CNS transplantation; neurodenerative diseases; Alzheimer's disease; Parkinson's disease; Huntington's disease; amyotrophic lateral sclerosis; HIV-related encephalopathy; and "stiff-man" syndrome.

33. The method according to claim 24, wherein said immune privileged site is the eye.

34. The method according to claim 33, wherein said inflammation is associated with a disorder selected from the group comprising: uveitis; conjunctivitis; chorioretinitis; uveoretinitis; optic neuritis; intraocular inflammation, such as retinitis and cystoid macular edema; sympathetic ophthalmia; scleritis; retinitis pigmentosa; inflammatory components of degenerative fondus disease; inflammatory components of ocular trauma; ocular inflammation caused by infection; proliferative vitreoretinopathies; acute ischemic optic neuropathy; excessive scarring, for example, following glaucoma filtration operation; and inflammation reaction against ocular implants.

35. The method according to claim 24, wherein said immune privileged site is the testis.

36. The method according to claim 35, wherein said inflammation is associated with a disorder selected from the group comprising: orchitis; epididimo-orchitis; infertility; and orchidal trauma.

37. The method according to claim 23, wherein said animal is a mammal.

38. The method according to claim 37, wherein said mammal is a human.

39. A method of creating an immune privileged site in a tissue of an animal comprising administering an effective amount of Fas ligand fragment comprising the extracellular domain of a full length Fas ligand, or a derivative thereof, behind the blood-tissue barrier of the tissue, wherein said Fas ligand fragment, or derivative thererof, has the ability to induce apoptosis in Fas expressing cells.

40. The method according to claim 39, wherein said animal is a mammal.

41. The method according to claim 40, wherein said mammal is a human.

42. A method of modulating inflammation in an immune privileged site in an animal through the *in vivo* induction of apoptosis in Fas expressing cells, comprising introducing an effective amount of a Fas ligand fragment comprising the extracellular domain of a full length Fas ligand, or a derivative thereof, behind the blood-tissue barrier of the immune privileged site.

43. The method according to claim 42, wherein said animal is a mammal.

44. The method according to claim 43, wherein said mammal is a human.

45. A method of modulating inflammation within an immune privileged site in an animal comprising the steps of:

(a) transforming cells *in vitro* with a nucleic acid encoding a Fas ligand fragment comprising the extracellular domain of a full length Fas ligand;

(b) selecting the cells transformed in step (a) that express the Fas ligand fragment;

(c) introducing the cells selected in step (b) behind the blood-tissue barrier of the immune privileged site,

(d) wherein said Fas ligand fragment, or derivative thereof, has the ability to induce apoptosis in Fas expressing cells.

46. The method according to claim 45, wherein said animal is a mammal.

47. The method according to claim 46, wherein said mammal is a human.